# Cardiac Output (CO)

ILOs:

By the end of this lecture the students will be able to:

1. Define stroke volume, cardiac output, and cardiac index
2. Explain the influence of altered preload on force of contraction of the cardiac muscle.
3. Describe the factors that help in venous return in upright position
4. Explain the role of contractility and after load in regulation cardiac output.
5. Describe the Indices of myocardial contractility.
6. Apply the information studied in this section to solve a clinical problem or explain clinical case.

## Definition:

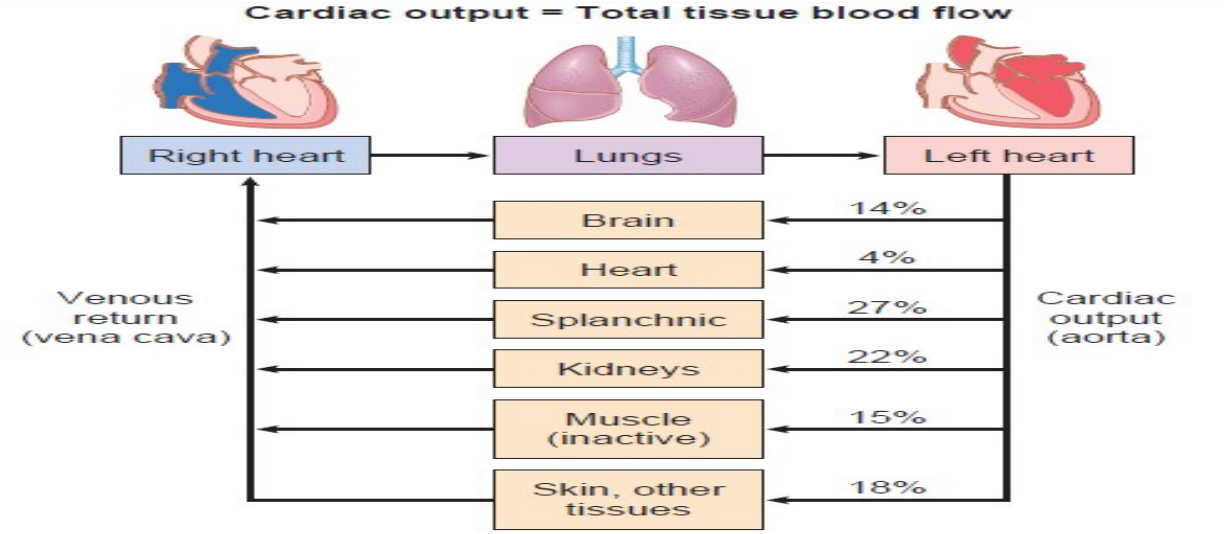
- ✓ Cardiac output is the amount of blood pumped from each ventricle in one minute.
- ✓ It equals 5 L/min in adult healthy man in supine position.

## Factors affecting CO :

Cardiac output varies widely with:

1) The basic level of body metabolism.
2) The level of activity of the body.
3) Exercise
4) Age
5) The body size.
6) Pregnancy
7) Body position standing or sitting.

For young, healthy man, resting cardiac output averages about 5L/min.




**Distribution of CO on different body organs**.
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**Definitions:**

**Stroke Volume:**

- ✓ It is the amount of blood pumped by the ventricle in one beat. In adult healthy man, it is equal to 70 ml blood in supine position.
- ✓ It is the difference between end systolic volume and end diastolic volume.

**End Diastolic volume**

- ✓ It is the amount of blood present in each ventricle at the end of diastole.
- ✓ It equals 130 ml and affected by venous return.

**End Systolic volume:**

- ✓ It is the amount blood remaining in each ventricle at the end of systole.
- ✓ It equals 60 ml and affected by arterial blood pressure and cardiac contractility.

**Cardiac Index :**

- ✓ The cardiac index is the cardiac output per square meter of body surface area.
- ✓ The normal average cardiac index for adult healthy man is about 3 L/min/m2 of body surface area.

**Ejection Fraction:**

- ✓ It is the ratio of stroke volume to end diastolic volume. It averages 60%.

**Measurement of Cardiac output:**

- ✓ can be measured noninvasively with Doppler echocardiography.
- ✓ velocity of blood in the ascending aorta is measured, and knowing the cross-sectional area of the aorta (also measured by echocardiography), we can determine stroke volume.
- ✓ Cardiac output is calculated by multiplying stroke volume by the heart rate.

**Factors determining CO:**

Co = Stroke volume (SV) × Heart rate (HR)

From the previous equation we can say that CO is determined by:

a) Stroke volume :
   - ✓ It has direct relationship with CO (CO increase by increased SV).
   - ✓ it is affected by preload ( venous return), afterload ( arterial blood pressure) and myocardial contractility.

b) Heart rate:
   - ✓ It has direct proportion with CO, if the stroke volume is kept constant.
   - ✓ It is affected by autonomic nerves.

**Factor regulating CO:**

**1) End Diastolic volume (*EDV*) (Preload, venous return, Heterometric regulation):**

- ✓ **Stroke volume is increased with increased EDV due to: Frank- Starling's law** of the heart states that the "energy of contraction is proportional to the initial length of the cardiac muscle fiber within limits". The length of the muscle fibers is proportional to the end-diastolic volume.
- ✓ In addition, the stretch of the sinus node in the wall of the right atrium by the coming blood, increase the heart rate ( directly and through Bainbridge reflex) so the CO increase.

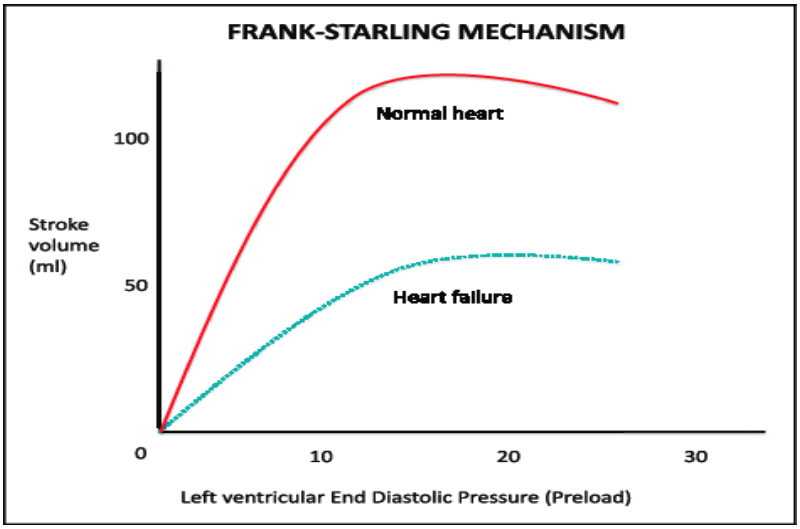
✓ **End diastolic volume is affected by :**

a) Venous return ( increased by exercise, increased negative intrathoracic pressure)

b) Systolic contractions of the ventricle ( weak contraction increase the EDV).

c) Diastolic functions as increase in intrapericardial pressure limits the ventricular filling (eg, as a result of infection or pressure from a tumor)

d) The total blood volume (Direct relationship): decreased blood volume decrease the venous return and CO. while decreased blood viscosity, increase the venous return and CO.




https://www.researchgate.net/profile/Mitali_Sahni2/publication/282437873/figure/fig1/AS:797638790246401@1567183315869/Effect-of-changes-in-myocardial-contractility-on-the-Frank-Starling-curve-The-curve.png

***Factors affect the venous return to the heart:***

*1) Venous pressure gradient:*

- Difference between right atrial pressure (RAP) and systemic filling pressure (SFP).
- Venous return increase with increased pressure gradient(= decreased RAP and increased SFP).
- In supine position it equal about 10 mmHg.
- Cardiac pumping help to keep RAP 0 mmHg.

2) *Muscular pump*:

- Muscle tone help to maintain venous return
- Contraction of the  during exercise  help to push more blood toward the heart.

*3) The inspiratory pump:*

- During inspiration the negative intrathoracic pressure help venous return.
- Its effect increase by deep breathing.

*4) Sympathetic tone:*

- Cause venoconstriction keeping the SFP higher than atrial pressure and help venous return

*5) The valves of the vein:*

- Keep the blood pushed in direction of heart and doesn't return with gravity.

*6) Capillary tone.*

- Only 10% of the capillaries is opened . If all of the capillaries are opened, the blood will accumulate in it preventing venous return.

**2) Myocardial contractility:**

- ✓ Regulation due to changes in contractility independent of length is sometimes called **homomeric regulation.**
- ✓ The contractility of the myocardium is directly proportion to stroke volume and CO.
- ✓ Contractility is affected by :

| +ve inotropic factors (↑↑) | -ve inotropic factors (↓↓) |
|---|---|
| The sympathetic nervous system (β1) | The parasympathetic nervous system ( M2) |
| Circulating catecholamines (β1) | Intrinsic depression (heart failure) |
| Digitalis ( inhibition of $Na^+$-$K^+$ pump). | Loss of myocardium |
| Increased heart rate due to increased availability of intracellular $Ca^{2+}$. | Hypoxia and acidosis |

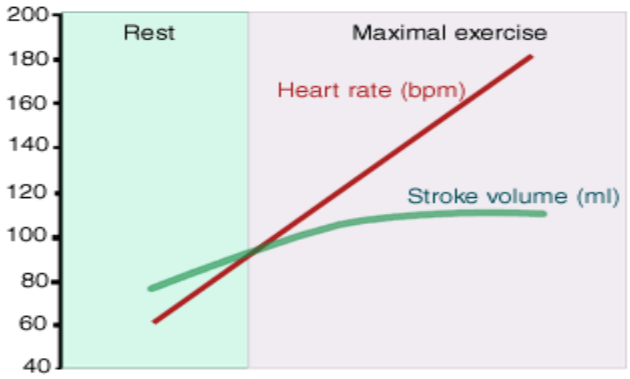
**3) Effect of peripheral resistance on CO:**

- ✓ The cardiac output has reciprocal relationship with changes in total peripheral resistance ( with unchanged arterial pressure).

$$\text{Cardiac output} = \frac{\text{Arterial pressure}}{\text{Total peripheral resistance}}$$

**4) Effect of heart rate on CO:**

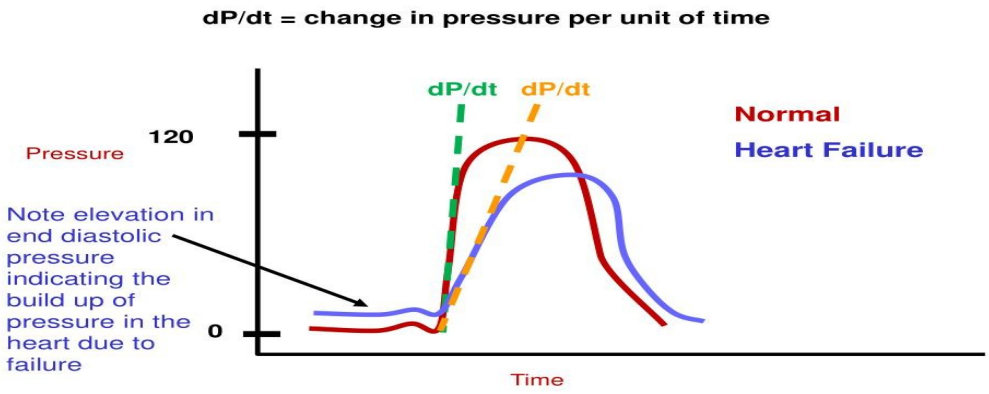
- ✓ Heart rate has direct proportion to cardiac output ( with constant SV)
- ✓ Heart rate is increased by sympathetic stimulation (+ve chronotropic effect) and decreased by parasympathetic simulation (-ve chronotropic effect).
- ✓ Very rapid heart rate will decrease CO : as increased heart rate will decrease filling time, ventricular filling and decreased SV.




https://www.medicine.mcgill.ca/physio/vlab/exercise/img/heartrate_strvol.gif

**Indices of myocardial contractility :**

1) dp/dt ( change in pressure versus change in time) : rate of development of the pressure during isovolumetric contraction phase.
2) Ejection Fraction (EF): it is percent of blood ejected from the ventricle and it give information about contractility. EF less than 40% indicates heart failure. It can be measured by echocardiography.




**Indices of contractility**

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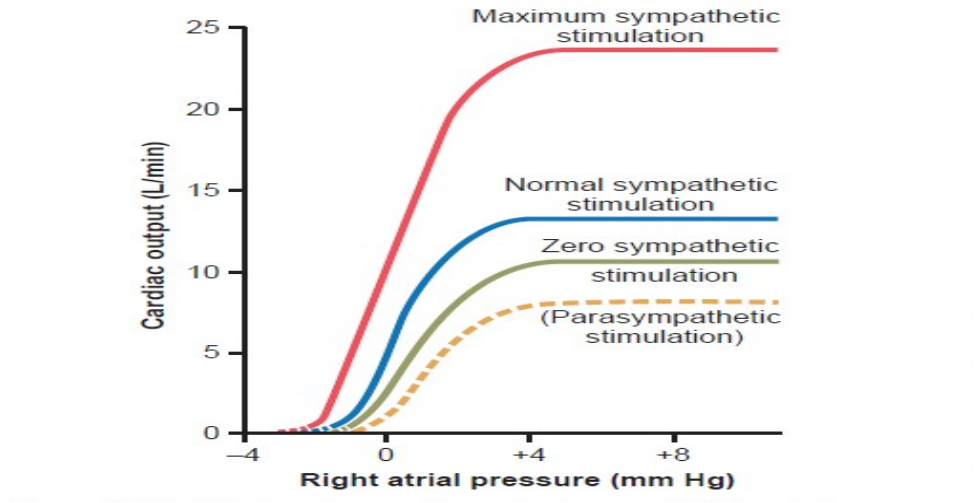
**Integrated control of cardiac output:**

The different mechanisms affecting CO operate together to maintain Co enough for body needs. Let's apply this to :

1) Heart during exercise:
   - During exercise sympathetic stimulation increase heart rate, increase contractility, increase stroke volume and CO . This is augmented by circulating catecholamines.
   - Muscle contraction during exercise and increase respiratory rate increasing venous return, end diastolic volume and CO.
   - Due to vasodilation of skeletal muscle blood vessels, peripheral resistance and after load are decreased and consequently CO is increased.

2) In patients with transplanted heart during exercise (denervated heart):
   - Heart operates according to Frank- Starling's law as venous return is increased with increased filling and end diastolic volume and increased CO
   - Circulating catecholamines share in increased CO.

3) Increased sympathetic activity to the heart causes :
   - Decrease systolic interval by increasing contractility and increased peak ventricular pressure.
   - Decrease diastolic interval by increasing heart rate: as intracellular calcium is increased more than sequestrated calcium. So contractility increase and compensate for decreased filling.




Effect of sympathetic stimulation of the heart on CO
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